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What is the Minimum Volume of Aerobic Physical Exercise Necessary to Elicit Postexercise Hypotension?

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ABSTRACT

Barreto CB, Aguiar SS, Palmeira R, Coelho Junior HJ, Gargaglione EML, Oliveira JF, Pires FO, Asano RY. What is the Minimum Volume of Aerobic Physical Exercise Necessary to Elicit Postexercise Hypotension? **JEPonline** 2015;18(6):1-12. Benefits from physical exercise are not restricted to a chronic stimulus. They can also be observed after a single session of exercise (such as the phenomenon called postexercise hypotension, PEH). While moderate aerobic exercise is known to cause PEH, the minimum exercise volume to elicit this phenomenon is not well elucidated. Twenty-two normotensive sedentary healthy adults were allocated to two groups: the Control Group (CG, n = 9) and the Exercise Group (EG, n = 13). The EG was randomly assigned to three aerobic exercise protocols with different volumes (10 min [S10], 20 min [S20], and 30 min [S30]) at 60% of heart rate reserve. Before and during the 30 min after the exercise bout, blood pressure was recorded. Results showed that moderate aerobic exercise sessions lasting 20 and 30 min significantly decreased the systolic blood pressure throughout the period after the exercise bout (S20: P<0.0001, ES15' = 1.3, ES30' = 1.6; S30: P<0.0001, ES15' = 1.1, ES30' = 1.4), Hence, the minimum exercise time to observe PEH after engaging in moderate aerobic exercise is 20 min.

Key Words: Hypotension, Aerobic exercise, exercise volume, Post exercise

INTRODUCTION

Hypertension is a major public health problem worldwide. In 2000, one billion people were diagnosed with this pathological state, but projections for 2025 indicate that this number will increase to 1.45 billion (24,46). The anticipated 29% increase in hypertension will contribute to the present pandemic of cardiovascular diseases (e.g., acute myocardial infarction) and early deaths worldwide. Hence, the world health organization (WHO) has characterized the increased prevalence of hypertension as the main factor of death in the world (43,53).

Recent meta-analytic data from randomized clinical trials indicate that engaging in regular aerobic physical activity can help reduce the burden of high blood pressure related complications in hypertensive and normotensive subjects (13). In agreement with these positive results, physical exercise is recommended as a powerful tool in the promotion of a positive and healthy lifestyle (1,34).

Although chronic physical exercise has been showed to have a positive effect of increasing lifespan, the health benefits are not restricted to chronic exercise. In fact, positive benefits to health can be observed from the first session of exercise (3,5,29). In fact, acute physical exercise has be shown to decrease the postexercise blood pressure response, which is a phenomenon called “postexercise hypotension” (PEH) (3,5,9,18,20,29,35,36,41).

Among the different types of exercise, moderate aerobic exercise is a safe and documented intervention that effectively produces PEH (9,26-28). However, the minimum amount of stimulus, that is, the minimum exercise time of a moderate aerobic exercise bout that may elicit PEH is not clear. Considering that exercise protocols ranging from 15 to 60 min have been suggested to produce PEH (9,26-28) and that the time spent during the exercise sessions may be the most limiting factor to the individuals’ adherence to an exercise training program (4,42), a study that determines the PEH response to a moderate aerobic exercise with different durations may to better understand the development of PEH. Thus, the purpose of this study was to analyze the contribution of a moderate aerobic physical exercise bout with different durations in normotensive individuals on PEH responses.

METHODS

This study was developed in accordance with the Declaration of Helsinki (Resolution 196/96 of the National Health Council). All subjects signed a free consent form that explained the research objectives and all risks with the experimental procedures used in the study. The study’s protocol was previously approved by the Ethics Committee of the University of Mogi das Cruzes (n° 999.106).

Subjects

This study was based on a randomized-crossover design of 22 normotensive sedentary healthy male (n = 8) and female adults (n = 14) (26.5 ± 9 age; 22.2 ± 7 $kg \cdot m^{-2}$) who were allocated to two groups: the Control Group (CG, n = 9) and the Exercise Group (EG, n = 13). As to criteria the required to participate in this study, each subject had to be free from: (1) the “obese” classification using body mass index (BMI) (≥30 $kg \cdot m^{-2}$); (2) taking hormonal therapy, drugs and/or alimentary supplements; (3) smoking habits; or (5) any kind of cardiovascular (e.g., myocardial acute infarction), metabolic (e.g., type 2 diabetes mellitus), pulmonary (e.g.,

chronic obstructive pulmonary disease), neurological or psychiatric (e.g., schizophrenia), and musculoskeletal disease. The inclusion criteria required full attendance to the experimental sessions.

All subjects were instructed to refrain from physical exercise for 96 hrs before the tests, drinking coffee, alcoholic, and energy drinks during the 24 hrs before the tests. Although alimentary ingestion was not controlled, subjects were instructed to repeat the same breakfast in all experimental sessions. All tests were conducted between 7:00 and 10:00 am under controlled temperature (22º C).

Procedures

Individuals were asked to visit the laboratory in 4 sessions. The first visit was used to clarify the study protocol and to get the individual's agreement to participate in the study. Thereafter, the subjects completed a sociodemographic (e.g., marital status) and health status (e.g., diseases) questionnaire, and were evaluated for body composition. Then, the subjects were randomly assigned to the Control Group (CG) or the Exercise Group (EG).

To avoid cardiovascular risk to the subjects, pre-exercise resting blood pressure levels (BP) could not exceed a systolic blood pressure (SBP) of 160 mmHg and a diastolic blood pressure (DBP) of 100 mmHg. Also, if during exercise blood pressure was ≥250 mmHg for systolic or increased 12 mmHg after the warm-up, the exercise protocol was stopped (29).

Assessment of Body Composition

A weight scale with stadiometer Filizola® (Brasil) was used to determine body mass (kg) and height (cm). Body mass index (BMI) was determined by using the formula: body mass (kg) / (height [cm])^2. All subjects showed normal weight classification (34).

Determination of Heart Rate Reserve (HRres)

Heart rate reserve (HRres) was determined by using the formula: maximal heart rate (HRmax) – resting heart rate (HRrest) (24). A cardiac monitor (Polar® FT1, Finland) was used to record HRrest, which was determined while the subjects remained sitting in a quiet room for 15 min. Light and temperature were automatically controlled. HRmax was determined by using the formula: 205 - (0.42 x age), as proposed by Sheffield and colleagues (38).

Aerobic Physical Exercise Protocol

The EG performed three moderate cycling aerobic exercise bouts with intensity set at 70% of the HRres (Movement® LX.130, São Paulo, Brasil). The duration of each aerobic exercise bout was changed in order to provide different aerobic exercise volumes. The subjects cycled at 70% of the HRres for 10 (S10), 20 (S20), and 30 min (S30). The aerobic exercise bouts were performed in a random order with a minimal interval between them of 6 days. A cardiac monitor (Polar® FT1, Finland) was used to monitor the subjects' HR, thus ensuring that each subject maintained the required intensity.

Control Group (CG)

The subjects in the CG remained sitting for 65 min as follows: (a) 15 min for the hemodynamic parameters to stabilize; (b) 20 min to mimic the mean exercise time of the EG; and (c) 30 min to record hemodynamic parameters.

Measurement of Hemodynamic Parameters During and After Exercise

All procedures for the measurement of blood pressure were adapted from the VII Joint National Committee of High Blood Pressure (JNC7) (26). Before, immediately after, and during the 30 min after the end of the aerobic exercise session (15 [rec15] and 30 min [rec30]), the subjects remained quietly sitting with their feet on the floor in a room with light and temperature automatic controlled. Each subject's arm was positioned at heart level during the blood pressure measurement.

An appropriate-sized cuff was placed on the left arm, approximately at the mean point (heart level). The size of the arm cuff was selected after measuring the arm circumference (Sanny, São Paulo, Brazil). Blood pressure was recorded by the auscultatory method using a stethoscope and sphygmomanometer (Premium®, Brasil). Phases I and V of the Korotoktof sounds was used to determine systolic blood pressure (SBP) and diastolic blood pressure (DBP), respectively. An experienced evaluator performed all measurements. The mean arterial pressure was determined using the formula: [SBP + (2 x PAD)] ÷ 3 (12,40).

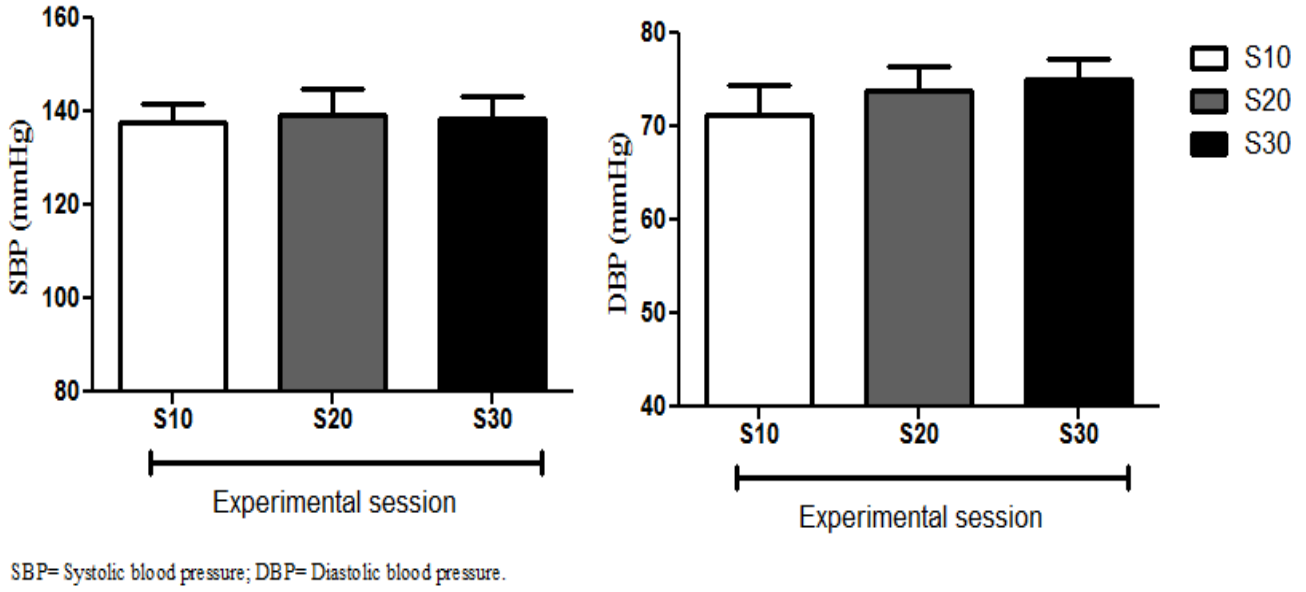
Statistical Analyses

The Shapiro-Wilk test was used to calculate data normality. Comparison between (CG and EG [S10, S20, and S30]) and intra-groups (Rest, 15' and 30') were performed by Split-Plot ANOVA, having a Bonferroni test as *post hoc*. Effect size were definite to be medium for values for Cohen's (**d** of more than 0.2 but less than 0.5, good for values between 0.5 and 0.8 and large for values ≥0.8).

The significance was set at 5% ($P<0.05$) in all analysis using the Statistical Package for the Social Sciences (New York, USA) and G*Power software version 3.1.9.2. Based on the magnitude of mean difference for each dependent variable (SBP, DBP, and MAP), the calculated sample size of 22 subjects would be required to provide power of analysis (β) ≥0.80 at $P=0.05$.

RESULTS

Figure 1 shows the cardiovascular responses during the experimental sessions (i.e., S10, S20, and S30) in EG. There were no significant differences in blood pressure values when measured immediately at the end of the exercise sessions of different durations (SBP: $F=0.03$, $P=0.96$; DBP: $F=0.45$, $P=0.63$).



SBP= Systolic blood pressure; DBP= Diastolic blood pressure.

Figure 1. Cardiovascular Responses in the Experimental Sessions.

Figure 2 shows the behavior of SBP before and during the 30 min after the end of the exercise in EG and CG. When compared to the rest pre-exercise period, the SBP showed a significant decrease throughout the S20 and S30 exercises, but not in S10 exercise and CG for the same duration (S20: $F=23.33$, $P<0.0001$, ES15'= 1.3, ES30'=1.6; S30: $F=18.19$, $P<0.0001$, ES15'=1.1, ES30'=1.4). Regarding the PEH, the magnitude of the decrease in SBP in S20 and S30 exercises was higher than in the CG ($P<0.01$ in both groups) during the 30 min of recovery (ES S20=1.4; ES S30=1.7). However, only the S30 exercise showed a decrease in SBP, significantly greater than in S10 exercise throughout the post-exercise phase (ES 15'=0.6 and ES 30'=1.7; $P<0.05$ to both moments).

Importantly, the S30 exercise bout was the only session that showed a significant decrease in DBP at the 15 and 30 min of the post-exercise phase, in comparison to the rest pre-exercise period ($F=10.20$, $P<0.0007$, ES 15'=0.7 and ES 30'=1.2). Thus, there were no significant differences in SBP between the other aerobic exercise sessions (Figure 2b).

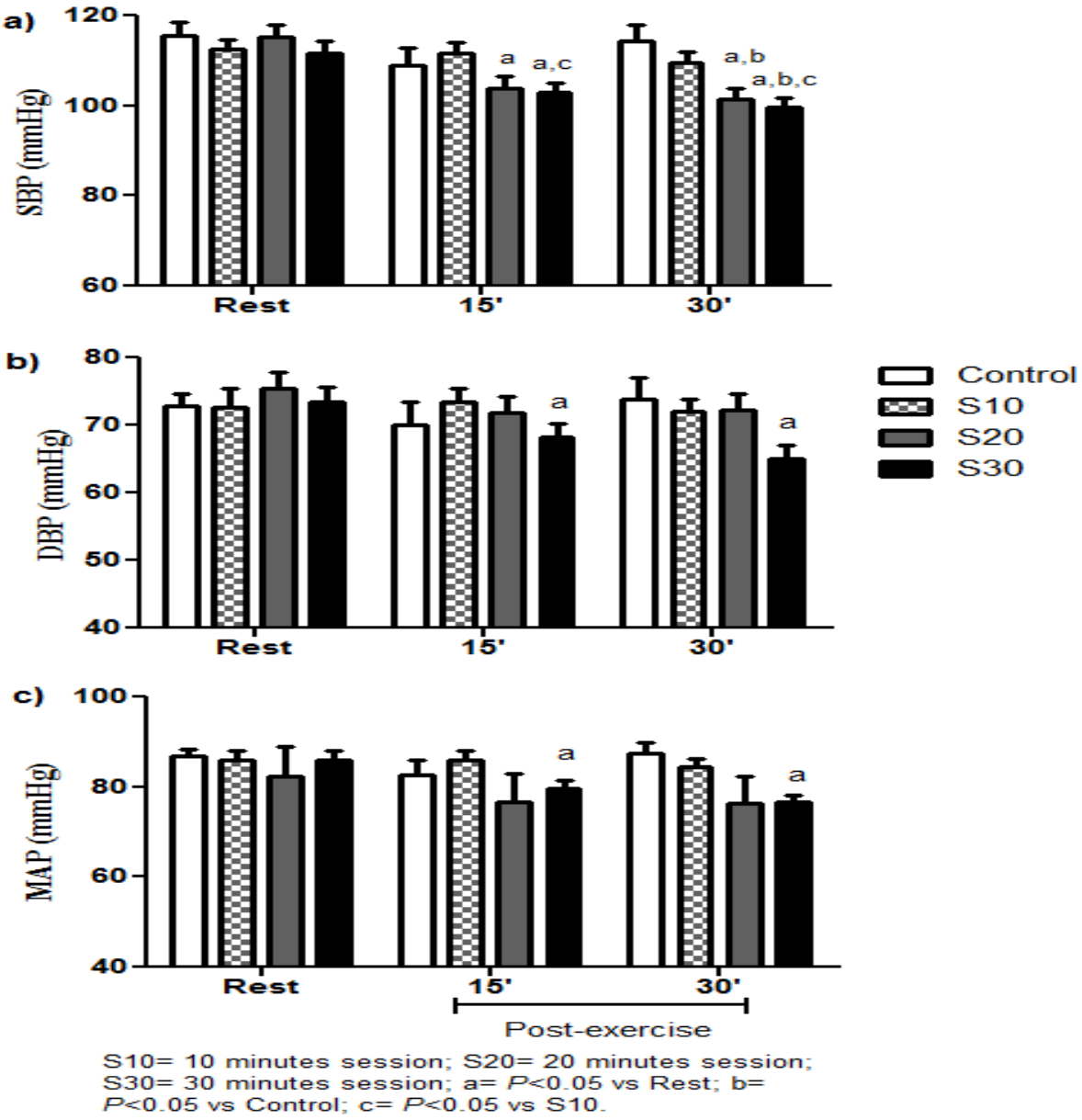


Figure 2. Hemodynamic Behavior after the End of the Experimental Sessions.

Accordingly, a significant decrease in MAP was observed only after the S30 exercise, throughout the post-exercise period (F=17.44, P<0.0001, ES 15'=0.9, and ES 30'=1.3). There were no significant differences between the other aerobic exercise sessions (Figure 2c).

DISCUSSION

The findings in the present study indicate that a single bout of aerobic moderate-intensity exercise lasting either 20 min or 30 min elicits the PEH response. However, this was not the case with the single bout of aerobic moderate-intensity exercise lasting 10 min. The 30-min exercise session (S30) may show SBP decreases than shorter aerobic exercise bout

sessions (S10). Moreover, 30-min exercise session (S30) was the only exercise duration that elicited PEH (based on the DBP and MAP responses) throughout the post-exercise recovery.

Although the evidence is conflicting, given the physical training configuration of volume and intensity, the magnitude of the PEH response in normotensive and hypertensive subjects may vary (7,9). Theoretically, while longer exercise sessions with greater volume of work are likely to result in greater decreases in blood pressure (9), there is no consensus if exercise volume affects the hypotension effect.

MacDonald et al. (27) did not find significant differences in the magnitude of PEH in normotensive men who underwent three moderate aerobic exercise protocols with different volumes (15, 30, and 45 min). However, 1 hr after the exercise sessions had been completed SBP started returning towards the resting values before exercise in the 15-min and 30-min exercise protocols. This was not the case in the 45-min exercise session in which the blood pressure remained decreased. These results were corroborated by Guidry et al. (19) and Jones et al. (22). They, too, did not observe a relationship between the exercise volume and the magnitude of the PEH. On the other hand, Forjaz et al. (17) reported PEH responses that were related to the exercise duration of which their subjects were similar to the subjects used in the present study,

In the present study, the longer exercise sessions (i.e., S20 and S30) were more effective in eliciting PEH (as noted in the SBP, DBP, and MAP responses) than the shorter exercise exercise (i.e., S10). Furthermore, there was a greater decrease in SBP after the S30 exercise, when compared to the other exercise durations. Differences between the results of the present study and the aforementioned studies may be explained by the method used to set the exercise intensity. Similar to the Forjaz's and colleagues' (17) study, the present sample was composed of healthy, sedentary young volunteers. In fact, the blood pressure responses prior to the exercise and the physical fitness status (e.g., sedentary) may have influence the PEH responses after the exercise bout (20,30-32).

The threshold to activate or increase the concentration of vasoactive substances, which is necessary to elicit a decrease in blood pressure, may require a minimal time interval of exercise (17). In the present study, the longer sessions (S20 and S30) were able to elicit SBP PEH; whereas, only the S30 exercise showed a decrease in DBP. Alterations in SBP and DBP PEH have been explained by the decrease in cardiac output (Q) and peripheral vascular resistance (PVR) after the exercise, but without compensatory increase in peripheral vascular resistance (PVR) during the recovery phase (36).

On the other hand, studies which showed SBP PEH, but without DBP PEH were associated with a compensatory increase or the absence of changes in PVR after exercise (36). Therefore, two explanations may be suggested to explain our results: (a) the longer physical exercise duration was sufficient to increase the vasoactive substance concentration (e.g., which has high release threshold) and/or enable/disable nervous system that can influence Q and PVR, causing SBP and DBP to decrease; and (b) the longer physical exercise duration was able to elicit an increase in other vasoactive substances and/or enable/disable nervous system, thus leading to a simultaneous biological activity of blood pressure controls.

Because the nervous system regulates cardiac chronotropism and inotropism as well as vascular tone, it can also regulate SBP and DBP. Sympathetic cardiovascular regulation is affected by arterial and cardiopulmonary baroreflex, which act as afferent pathways from peripheral environment. During physical exercise, there is a shift of baroreflex activity to a superior level in comparison with pre-exercise level (20). However, after exercise, baroreflex are reprogrammed to act in lower blood pressure levels. Yet, this effect is predominantly showed in hypertensive rats, and may not be observed in normotensive humans (33,46). Furthermore, some experiments observed increase in heart rate after aerobic physical exercise, which could limit the inferences of nervous system as a possible mechanism to explain the results of the present study (8,15,17).

Physical exercise is a means to increasing vasoactive substances bioavailability (e.g., bradykinin and nitric oxide [NO]), which can cause arterial vasodilation (2,3,5,37,41) as well act on the cardiovascular central control and in the cardiomyocyte (6,16). The NO is a key factor in arterial vasodilation regulation (44), so that an increase in NO bioavailability is proposed as a trigger factor to PEH (2,3,5). In relation to central and cardiac control, some experiments (i.e., *in vivo* and *in vitro*) observed that NO inhibition can cause an increase in cardiac chronotropism, whereas increases in NO bioavailability reverses this phenomenon (6,16). In complement, NO inhibition in humans elicits an increase in R-R interval and thus a decrease in parasympathetic activity (11,33). It is possible that longer physical exercise may induce higher shear stress with a dose-dependent relationship with NO bioavailability (23).

Other molecular and cellular pathways can be activated by longer physical exercise and affect the PEH (e.g., release of metabolites, heat) (17). However, due to methodological limitations, the present study was limited in addressing possible mechanisms related with PEH and longer physical exercises. Another limitation of the present study was the methodology that set the exercise intensity, given that the present study used an indirect method based on the HR. This factor may have limited comparisons with previous results. However, given that the HRres method is a useful and simple tool in clinical practice and exercise interventions, the methodology should be considered highly applicable.

A recent study by Asano et al. (2015) presented evidence that the time spent in physical exercise practice is the most limiting factor of the adherence to physical exercise programs. Thus, the model of physical exercise used in this study constitutes a short and beneficial means to controlling blood pressure values. Future studies should include biochemical and neural modulator measures as well as longer exercise bout sessions (40, 50, and 60 min) in order to improve the understanding of the PEH model.

CONCLUSIONS

Moderate aerobic physical exercise sessions of 20 and 30 min in duration were capable of producing a significant decrease in postexercise systolic blood pressure in normotensive subjects. Future research in this area will clarify whether the minimum time to experience PEH after engaging in moderate aerobic exercise is physiologically linked to 20 min.

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